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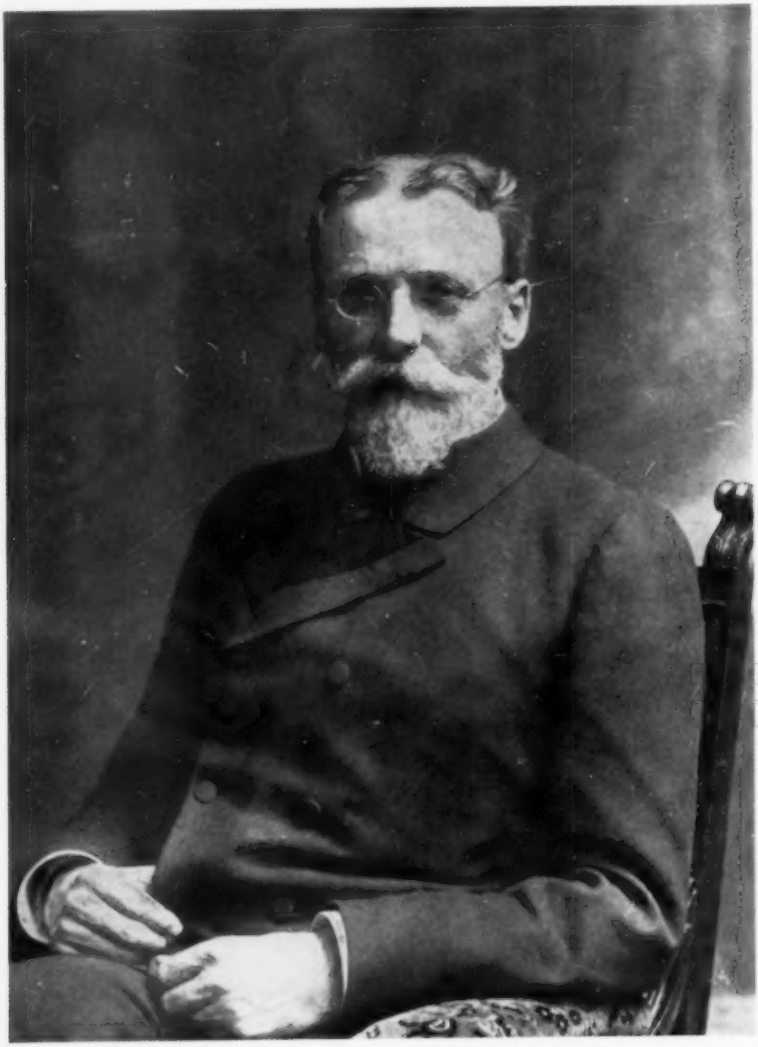

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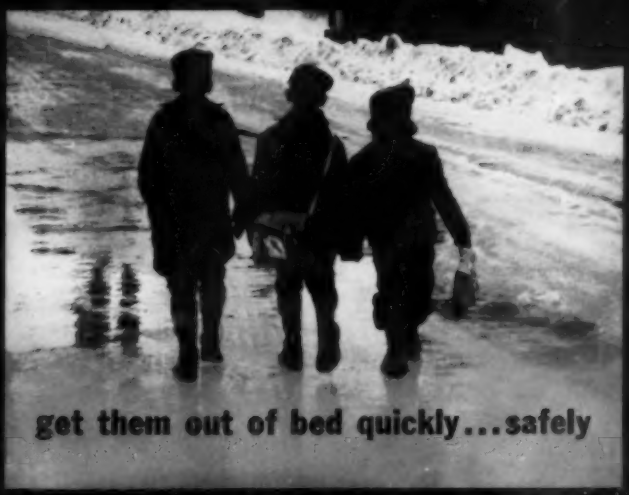

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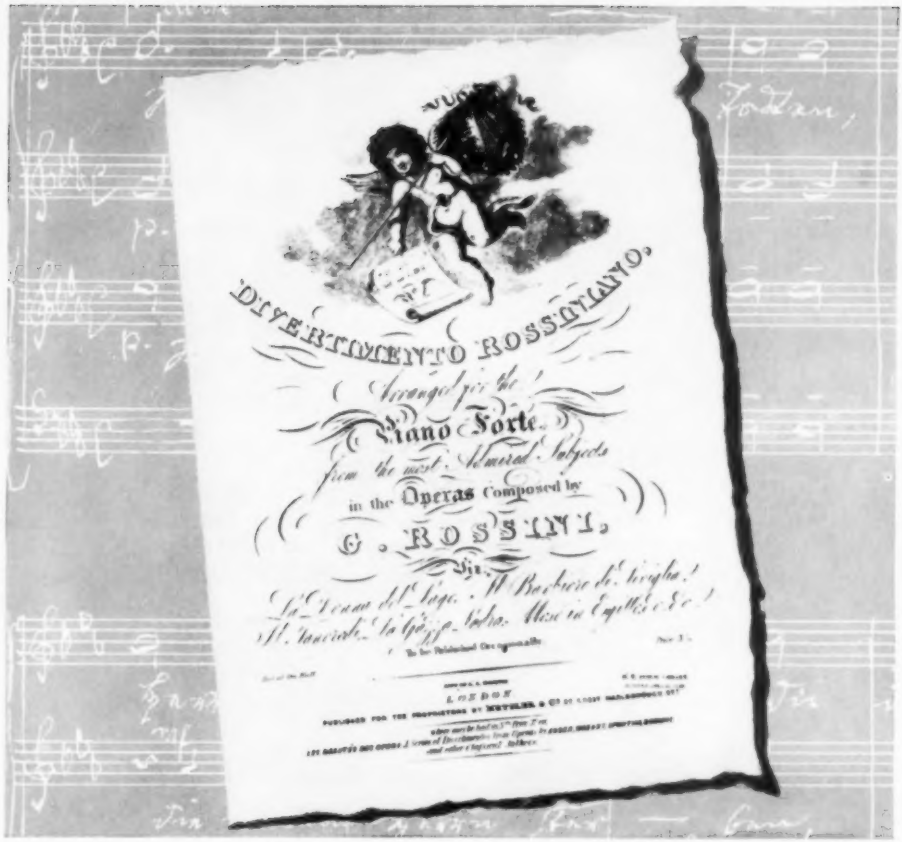

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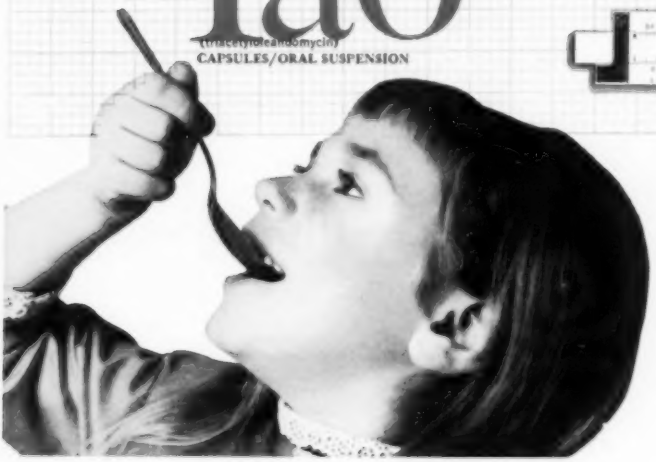

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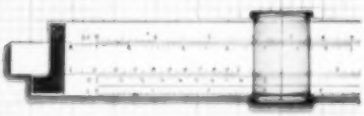

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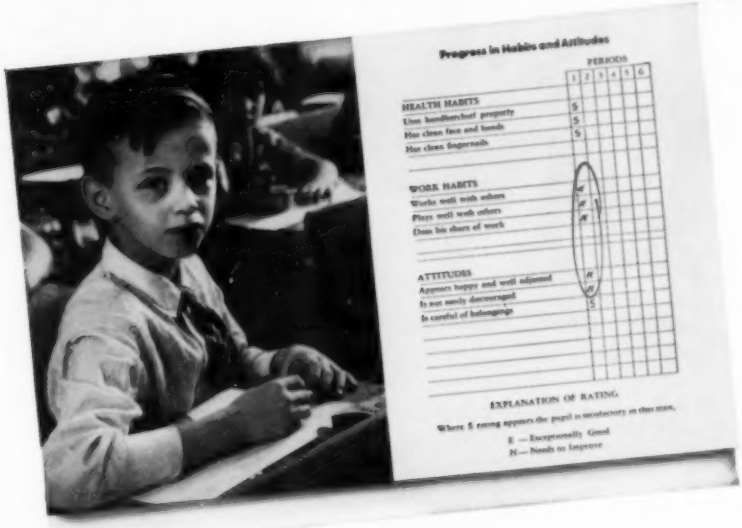

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Vol. 76 FEBRUARY 1959 No. 2

CHYLOTHORAX IN THE NEWBORN

DAVID J. DAHL, M.D.*

PHILIP N. SAWYER, M.D.*

New York

Chylothorax in the newborn is the collection of a chylous effusion within a hemithorax in the neonatal period. It is thought to be due to a tear in the thoracic lymphatic duct or one of its tributaries. The condition was first recognized and reported in the newborn by Stewart in 1926[1]. Eighteen cases have now been reported in the literature. In view of its rarity, an additional case report seems warranted.

CASE REPORT

The patient was a normal, full-term Puerto Rican male. The prepartum history and delivery were normal. There was no familial history of congenital defects or disease. Just prior to discharge from the hospital on his seventh post-partum day, he developed what appeared to be an upper respiratory infection. On admission to the pediatric service he was a normally proportioned, acutely ill child with a nasal mucous discharge, a rectal temperature of 100° F., a pulse rate of 120, and a respiratory rate of 46. History and physical examination were indicative of complete, or nearly complete collapse of the left lung by fluid, with mediastinal shift to the right. This was confirmed by x-ray. On thoracentesis, 120 cubic centimeters of orange-yellow, creamy fluid was aspirated from the chest. Wide spectrum oral antibiotics, aureomycin 5 mgs./kg./day and chloromycetin 20 mgs./kg./day were started. A smear of the thoracic aspirate revealed lymphocytes with a few mesothelial cells, suggesting that the material might be chyle. After the first thoracentesis the dyspnea disappeared; the child remained afebrile, and both lungs

* *From the Pediatric and Surgical Services of St. Luke's Hospital, New York, N. Y.*

expanded completely. In forty-eight hours, however, physical examination and a repeat chest x-ray demonstrated a re-accumulation of fluid. Five thoracenteses were performed at increasing intervals of time during the hospital stay of four weeks. The last was performed one month after birth. The child did not gain weight while he was accumulating chyle in the chest in spite of an excellent caloric intake, probably because of the loss of protein and fat (and the calories these substances represent) which were removed by thoracentesis. Diet became an important factor in treatment. Initially he received a formula of half skimmed milk (approximately 60 cal. per lb. per day). His diet was changed to evaporated milk on the sixteenth post-partum day in an attempt to increase caloric intake to 80 to 100 calories per pound per day. With the onset of diarrhea and a punctate erythematous rash on the forty-third day after birth, his diet was again changed to a high protein formula supplying 60 to 70 calories per pound per day. Between feedings he was offered glucose containing dilute amounts of sodium, potassium and other electrolytes. At the same time antibiotics were stopped. The seven to twelve loose, watery bowel movements a day continued. Electrolytes remained normal during the episode of diarrhea which lasted for seven days. The patient's weight began to increase during this period. The rash disappeared during the second day of the diarrhea, and at the end of the period of diarrhea it was found that the chylothorax had not re-accumulated. Follow-up examination at four months and six months of age revealed the child to be developing and gaining weight normally without re-accumulation of thoracic fluid.

LABORATORY DATA

The following table compares a few of the findings of our patient with those reported previously.

Tests on Thoracic Chyle	Everhart[2] Group	Sterlacci[3] Group	Authors' Case
Specific Gravity	1.012 – 1.018	1.012 – 1.016	1.010 – 1.016
Cell count lymphocytes	95 – 99%	—	99 – 100%
Fat total	0.9 – 4.8%	—	0.9 – 2.5%
Total cholesterol	—	—	48 – 60 mg.%
Lipid phosphorous	—	—	5.4 – 6.6
Total protein	3.0 – 8.0 gm.%	3.1 – 4.7 gm.%	2.5 – 4.2 gm.%
Albumin-globulin ratio	—	—	3.1/1.1
pH	7.4	Alkaline	7.4
Sudan four stain	Positive	Inconclusive	Inconclusive
Culture	Sterile	Sterile	Sterile
Test on blood serum			
Serum albumin-globulin ratio	—	—	4.1/0.9

DISCUSSION

The literature reveals that such infants are usually the product of a normal term pregnancy, and a normal labor and delivery. At first the child does well but then develops unexpected dyspnea, rib retraction, a rapid pulse, discomfort and a low-grade fever. Dullness is usually noted in the affected hemithorax with shift of the mediastinum to the unaffected side. If not recognized, the condition progresses until cyanosis supervenes. Death may occur if progression of the disease with further collapse of lung continues. An immediate thoracentesis usually reveals milky, non-purulent fluid within the thorax. Subsequent thoracenteses are frequently necessary.

Additional treatment can be resolved into (1) maintaining adequate nutrition, (2) control of diarrhea, and (3) maintaining adequate oxygenation. Thoracotomy is usually not indicated unless the infant becomes progressively worse while on conservative treatment. In ten of the eighteen patients reported in the literature, the chylothorax disappeared spontaneously. Randolph and Gross[4] suggest that conservative therapy without improvement be limited to twenty-one days. In five of the reported infants, thoracotomy was resorted to in an attempt to repair a rent in the thoracic duct or branches. In only two of the five patients was the ligation apparently successful.

Most of the chylous effusions have been sterile. In our case antibiotics were used prophylactically to prevent infection following thoracentesis. Everhart and Jacobs[2] make a distinction between chylous and chyliform effusions. They feel a true chylous effusion should have a high fat content, high cholesterol and a low lecithin concentration, to be distinguished from a chyliform effusion which they define as having a low fat, low cholesterol and high lecithin content. They feel that chyliform effusion may be the result of a breakdown product of cellular metabolism which may occur in infections or neoplastic growths. The properties which appear common to all chylous effusions are a lymphocyte count approaching 100%, a slightly alkaline pH, a specific gravity over 1.010, and sterile culture of the fluid. The protein albumin-globulin ratio of the removed chylous fluid showed a proportion similar to that in serum. The diarrhea, for some unexplained reason, as in other reported cases, heralded the resolution of the chylous effusion.

In previous reports[4,5] two principal pathological findings have been described at operation and autopsy. These are generalized weeping of the entire pleural surface and leakage of the thoracic duct from several points. Ligation of the thoracic duct might be

effective in these latter cases. It has been suggested that traumatic tearing may occur at birth with subsequent healing. This seems possible when it is remembered that the thoracic duct is long and fragile. Randolph and Gross[4] suggest that the defect may be congenital.

Four of the cases reported in the literature did not survive. One of these had been operated upon. The one patient that was operated upon and subsequently died had, in addition to chylothorax, chylous ascites and lymphedema, suggesting multiple lymphatic anomalies. The three other deaths were in the group cared for by thoracentesis and supportive therapy. The total mortality of the reported patients, including the present living case presented, is 21%.

SUMMARY

A case of chylothorax is presented. Diagnosis of this condition was made by thoracentesis and study of the obtained fluid. Laboratory findings are varied, but a high lymphocyte count, specific gravity above 1.010, sterility of the fluid on culture, and slight alkalinity indicate the nature of the fluid. The etiology is unknown in a great proportion of the cases. It would appear that the initial therapy should be conservative. However, surgery may be indicated if the patient does not show definite signs of improvement within several weeks.

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IDIOPATHIC PULMONARY ATROPHY

(Vanishing Lung and Unilateral Emphysema)

Piero Fornara, M.D.*

Italy

Much has been written in these last years, both in America and in Europe, about the localized forms of pulmonary emphysema. Their frequency appears to be increasing, especially in children. This increase, however, is apparent only in part.

In 1952, in collaboration with Potts and Holinger, Fischer[1] had written about lobar emphysema of children: "We are unable to explain the frequency with which we have recently observed this type of case and the rarity of previously reported similar cases". More recently, in a paper written in collaboration with Lucido and Lynxwiler, Fischer wrote[2] that "the apparent increase in the incidence is probably due to two factors. The first is the wider use of chest filming and fluoroscopy of the young infant; the second, as so often is true in medical practice, is the awareness of the possibility of this diagnosis being made by the radiologist, the pediatrician or the surgeon".

According to the above-mentioned authors, these forms are thus characterized: "The site and nature of the lung lesion is indicated by the finding in chest x-rays, a highly radiolucent lobe or entire lung field which still contains lung markings". These signs, however, are common to conditions of a different nature, so that Mayer and Rappoport[3] are correct in writing: "The various lung changes under discussion here are so often combined, that great confusion in nomenclature has resulted in such descriptive terms as cysts, bullae, pneumatoceles, cystic bronchiectasis, cystic emphysema, bullous emphysema, pneumocystis, vanishing lungs and honeycomb lung".

Among these various conditions, one has peculiar characteristics and deserves particular mention. This is the form which De Martini described in 1948 as "solitary lobar atrophy; in 1951, as "Idiopathic pulmonary atrophy", and in 1957 as "idiopathic pseudoemphysematous pulmonary atrophy". This form can be observed in children and grown-ups who usually do not present any particular respiratory trouble; in their history there are sometimes recurrent bronchial and pulmonary inflammations. The radiographic examination, either made during one of these catarrhal episodes, or casually,

* From the Pediatric Service of the Ospedale Maggiore of Novara (Italy)
Director: Prof. P. Fornara, M.D.

reveals a particular finding. De Martini described the following characteristics:

1) regional hypertransparency of the lung
2) Lack of pathology in the hilum
3) mediastinal shift (for the lower lobes)
4) accentuation of the markings of the other lung

The British authors call this form "abnormal transradiancy of the lung" (MacLeod[4]) or "unilateral emphyseme" (Dornhorst, Heaf and Semple[5], Belcher, Pattinson and Smart[6,7,8]). Among the French authors Galy, Bethenod and Bailly[9] and particularly Bailly[10] (whose monograph of 1957 contains 63 cases found in the literature and three cases observed by the author) speak of "giant malformative lobar emphysema". In Germany Heilmeyer and Schmid[11, 12], describing five cases in 1956, write that "no detailed observations of such cases have as yet been reported in Germany", and discuss their relations with the "vanishing lung" described by Burke[13] in 1937, and by Allison[14] in 1942, with the "cotton-candy lung" described by Crenshaw[15] and with the "idiopathic pulmonary atrophy" described by De Martini in 1948. Uehlinger[16], in the same years, described some similar cases (one of them he studied also anatomically) under the name of "vanishing lung, progressive pulmonary dystrophy".

This form must be distinguished from the "progressive bullous emphysema of the suckling", often improperly called "lobar", of which Fischer and collaborators, in the two already-mentioned papers, report all the American references. Many other authors wrote about the same form: in France Bailly and collaborators[9, 10], who call it "emphysème malformatif bulleux géant"; in England White, Jones and Temple[17], Holzel, Bennett and Vaughan[18], Nelson[19], Cottini and Myers[20]; in Italy Fontana and Panzironi[21], Gomirato-Sandrucci and Poletto[22], Fornara[23], Panizon[24] and Gavini[25]. In 1957, in a paper on the hypertransparent pulmonary areas of the child[26], I insisted on the necessity of clearly distinguishing the bullous post-infectious forms or pneumatoceles "sensu Duken", the dysembryoplastic cysts and the bullous emphysemas from the solitary lobar atrophy, referred to in the first paper of De Martini who in 1948[27] had already described a first group of 17 cases. Later on, in 1951, De Martini and Balestra[28] describing a second group of 20 cases, further illustrated this condition pointing out that on bronchography the radiopaque substance does not reach the terminal bronchi, but injects only some proximal branches, and angiopneumography shows the complete absence of vascularization of the pulmonary bronchi at their origin, causing the amputated appearance of the corresponding hilum.

De Martini, in 1957[29], wrote again about this "idiopathic pseudo-emphysematous pulmonary atrophy", dwelling upon the clinical and radiological characteristics of this "syndrome of regional pulmonary rarefaction of atrophic type". The available data, he declared, are not yet sufficient to establish whether this syndrome is due to pulmonary hypogenesis or to a hypoplasia of the lobar pulmonary artery.

The first of these hypotheses has been supported in Italy by Garbagni and Fazio. These authors, in 1956[30], described the case of a young man of 20, who, at the age of 4 had had a right lobar pneumonia. A radiograph had shown an evident hypertransparency of the right lung, which was considered as an emphysema. This was still unvaried 16 years later; the bronchovascular shadows were not very pronounced and limited to the hilum, which was smaller than on the opposite side. The bronchographic, bronchospirometric and functional findings were reduced. The authors therefore think this syndrome is due to a congenital hypoplasia of the right lung, with hypoplasia of the corresponding bronchia and arteries. It is not clear what the authors mean—from the clinical point of view—by pulmonary hypogenesis. In 1912 P. Schneider[31], in his chapter of Schwalbe's text-book, made this distinction:

pulmonary agnesia is a complete absence of one lung;
pulmonary aplasia is an absence of the lung parenchyma;
pulmonary hypoplasia if the bronchial primordium is present.

The lack of precision in the definition of pulmonary hypoplasia in Schneider's text allowed very different personal interpretations in the papers on the subject, particularly in those by Field[32], by L. Schneider[33] and in the same article by Garbagni and Fazio[30]. Breton and Dubois[34], in their book on the congenital malformations of the lung, wrote thus: "en résumé, cette conception classique des hypoplasies pulmonaires aux limites si flous, a permis à beaucoup d'auteurs d'y ranger des affections qui ne devraient pas s'y trouver, et qu'ils n'ont pas placées dans un cadre nosologique mieux déterminé".

On the other hand, according to what is observed in the various forms of pulmonary agenesia, it is usually admitted (Field, Breton and Dubois) that the following radiologic findings are required for the diagnosis of pulmonary hypoplasia:

1. opacity of a hemithorax or of part of it,
2. absence of a bronchial branch of bronchography and of the corresponding vessel on angiography.

Pulmonary opacity is described also in the condition that Mac-

Mahon[35, 36] calls "congenital alveolar dysplasia" and that he considers it a malformation similar to pulmonary hypoplasia, but Potter,[37], on the contrary, interprets it as caused by the "hyaline membrane with resorption atelectasis".

In such cases as the one of Garbagni and Fazio[30], we can ask ourselves whether the cause is a primary vascular lesion, i.e. the congenital absence of a principal branch of the pulmonary artery; this was so in the two cases (also presenting a hypertransparency of the corresponding hemithorax) studied by Steinberg, Dotter and Lukas[38], who collected ten other similar cases in the literature.

This hypothesis of a primary vascular lesion is especially supported now by the English authors, among them Belcher, Smart and Pattinson, who record that, of the cases published as "unilateral emphysema" most are due rather to anomalies of the pulmonary arteries than to primary pulmonary abnormality. The anomalies may take the form of hypoplasia of a whole pulmonary artery as in the case described by Smart and Pattinson[6]; or hypoplasia of a lobar artery, as in the three cases described by Belcher and Pattinson" [7].

Also in the patient described by Dornhorst, Heaf and Semple[5], who was treated by lobectomy, "the pulmonary vessels were smaller than normal". This was not demonstrable in a similar case described by Swyer and James[39] as "unilateral pulmonary emphysema". The patient was a six-year-old child who, since the age of six weeks, had recurrent bronchial troubles. On bronchography the right peripheral bronchial branches were not completely filled and ended with small ampullary dilatations; on angiography, only a small part of the principal branch of the right pulmonary artery was visible. On operation, the right pulmonary artery, though hypoplastic, appeared much less reduced than the angiography showed. In the pulmonary parenchyma, however, there were alveolar areas partly collapsed and partly emphysematous, mingled with vesicular and cystic formations, connected with ectasis and chronic inflammation of the bronchioles. There was also "a widespread obliteration of the peripheral lung capillaries" that the authors consider as connected with a 'functional insufficiency of the right pulmonary artery, secondary to the widespread lung disease of uncertain origin", but perhaps due to recurrent bronchitis.

De Martini, discussing this case, warns that the negative angiographical finding may be due to a functional lack of permeability and not to the real structural situation of the vessels in question. MacLeod[4], discussing the pathogenesis of his case, writes: "I have already commented on the small vascular shadows seen by radio-

graphy in these lungs. There is no evidence to support the suggestion that the lung changes follow a congenital hypoplasia or absence of one pulmonary artery or a developmental abnormality of the bronchial arteries". The same can be said of the case published by Madoff, Gaensler and Strieder in 1952. In the already-quoted case of Swyer and James, the alterations seemed to be due more to a functional insufficiency of the pulmonary artery, than to a primary congenital hypoplasia of the artery. Belcher, Smart and Pattinson are of a very different opinion. They recently wrote: "We feel that the term unilateral emphysema is misleading in these cases, as no emphysema may be present. Hypoplasia of the pulmonary or lobar artery is, we think, a better term".

Also Heilmeyer and Schmid state that in their cases "the extensive obliteration of the pulmonary arterial system, which could only be proved by x-ray examination, predominates" and that "it is likely that vascular obliteration precedes pulmonary atrophy: for this reason we believe that simultaneous occlusion of the bronchial or pulmonary arteries is a sine-qua-non for the development of atrophy. A thorough pathological examination of these cases is necessary for further elucidation of the condition. An inflammatory form of endoarteritis obliterans must be considered the most likely cause of the vascular occlusion". Crenshaw[15], studying the operation specimens of 50 cases of emphysema, had particularly pointed out the existence of beginning lesions of the bronchial vessels of the type of vasculopathia obliterans. In 1953 Cudkowicz and Armstrong[10] had described similar lesions in 18 cases of chronic bronchitis and emphysemas, studied postmorten. It is difficult to state whether these vascular lesions are primary or secondary. De Martini thinks that there is no evidence to support the hypothesis that vascular aplasia or hypoplasia is primary and determinant in the pathogenesis of these forms: as an example he quotes the case studied also anatomically by Uehlinger, in which there was accentuated pulmonary atrophy without noticeable alterations of the vascular walls.

Some authors think that this form also may be caused by the "check-valve mechanism", which is responsible for most of the bullous pulmonary forms. Lister[11] recently wrote: "Emphysema is never a primary disease but is always secondary. It is a state of over-distension of the terminal air sacs of the lungs by check-valve action in the small bronchi or bronchioles. A variety of conditions can cause this".

This check-valve mechanism has been pointed out in 33 out of 55 cases of giant malformative lobar emphysema reviewed by Bailly[10]; its cause may be intramural, mostly owing to those folds of the mucosa shown by Robertson and James[42] who observed "valvelike folds of mucosa in the lobar bronchus" in 2 cases out of 5; sometimes it may be mural, identifiable with malformations of the bronchial wall; a particular flaccidity of the bronchi has been described in one case by Shaw[43]; "malformation or deficiency of the cartilaginous ring of the bronchus" is observed in 20 of the 55 above mentioned cases studied by Bailly[10] as well as in 7 of the 9 cases of bullous emphysema published by Nelson[19] and in 2 of the 6 cases described in the first paper of Fischer and collaborators[1]. In their second paper, these write that "about one third of the reported cases of lobar emphysema have been secondary to an abnormality of bronchial cartilage".

How this may happen is explained by Raso[44] in a paper about the congenital factors in bronchial diseases. He writes that the incomplete growth of the layers composing the walls of the large bronchi causes a stenosis. The consequences of this stenosis, however, are different from those developing under different pathological conditions. Overstreet, Fergusson and Neuhauser, Evans and Cassinelli have well proved that the lack of cartilaginous rings causes the walls of the bronchi to be easily compressed; their lumen narrows and generally becomes a slit; a very serious pulmonary emphysema occurs, evidently owing to a check-valve mechanism.

Furthermore, various extramural causes have been described; they are mostly anomalies of the great vessels at the base of the heart, such as the ductus arteriosus of Botallo, anomalous lung arteries, etc.

As Bailly writes, the bronchial pathogenesis, which was the first to be considered, can explain only the cases more similar to bullous forms than to real emphysematous forms, to which the check-valve mechanism cannot be easily applied. The same can be said of the essential emphysema of the old, about which Borden, Sweany and Lipton[45] write: "Most British authors place emphysema under the heading of chronic bronchitis. Chronic bronchial infection is known to be accompanied by hypersecretion of mucus and by inflammation. It is held that these processes obstruct the bronchioles by a check-valve mechanism and lead to overdistension of the accompanying air sac. Most American scholars, on the other hand, hold the view that the primary disturbance is a loss of elasticity of the lung".

This holds still more for localized emphysemas. In fact, they cannot be directly determined by parietal causes, responsible for ventilation troubles, but by a primary alveolar lesion: "au niveau du parenchyme pulmonaire lui-même se trouve l'origine de l'affection", writes Bailly. This is also the opinion of De Martini, who, even in his first paper (1948), distinguished this form characterized by a lung rarefaction limited to a lobe, considered idiopathic and primary (the form called "non-obstructive emphysema" by the American authors, says De Martini) from the segmental emphysema derived from an obstruction, that is, with the marks of a process of alveolar atrophy, secondary to a preceding distension of the alveoli (the "obstructive emphysema" of the American authors).

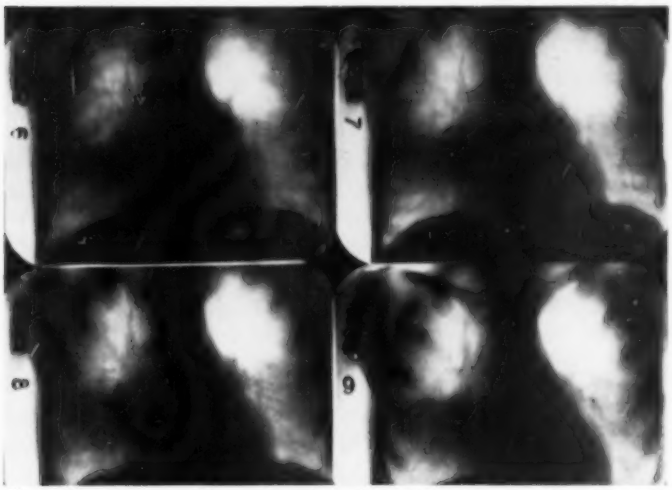

Fig. 1. Four tomographic sections through the lung fields showing increased radiolucency in the left field. May 28, 1956.

But what is the alveolar lesion and which is the cause? Some authors suppose the existence of a disease of the alveolar walls, strangely localized to a systematical region. Mayer and Rappaport[46, 47] speak of "a lung defect, mostly developmental in origin". Some authors (Letulle[48], Schmincke[49]) speak of a constitutional fragility of the alveolar system owing to dystrophy of the elastic tissue. Policard[50, 51] speak of constitutional hypoelastosis, Lantuejoul and collaborators[52] speak of parietoalveolar atrophy; Holzel, Bennet and Vaughan[18] think that there is a defect of the interstitial

tissue owing to hypoplasia or miopragia of some of its various elements, the reticular, the muscular or the elastic; Marie and collaborators[53] speak of a "lobar mega-alveolosis". Finally, other authors, such as Delarue[54, 55, 56] think that the alteration of the elastic system of the alveolar septa, provoking an "atrophic reticular pneumonia", may have as primum movens a nervous bronchial and peribronchial lesion (the "pneumolyse bronchogène réflexe" of Delarue).

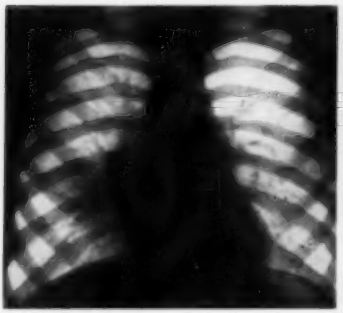

Fig. 2. Postero-anterior view. June 6, 1956.

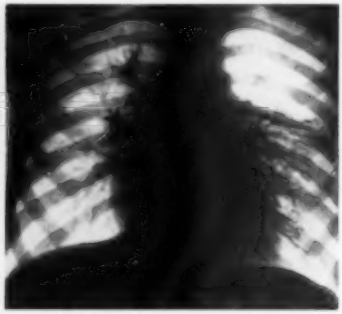

Fig. 3. Bilateral bronchograms. Note the poor filling of the finer bronchi in the upper lobe of the left lung. June 7, 1956.

Perhaps Bailly is right in writing that "des affections de nature intime différente sont classées sous cette même rubrique d'emphysème malformatif lobaire géant, et dès lors il n'y a pas lieu d'opposer les diverses interprétations et les types pathogéniques" and that "cet emphysème malformatif représente un syndrome recouvrant des faits différents". But, as De Martini writes in his last paper[29], the persisting impossibility of a precise etiogenetical classification, in addition to the clinical, radiological, bronchographic and angiographic data collected, keeps alive interest in this syndrome, which has prevailed until now in the diagnostic, prognostic and medicolegal field.

Also for this reason, and in order to stimulate further investigations, I present here a case that I have been following for years having all the characteristics of this form. I have already presented in my paper on the hypertransparent areas in children[26], contrasting it to other cases of bullous emphysema treated by exeresis of the bullae.

The patient is a male child who had pneumonia when one year old; when six, in May 1956, he was admitted to my department for

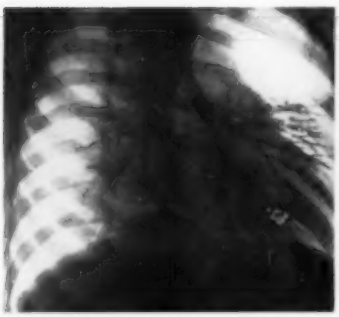

Fig. 4. Oblique bronchographic view. June 7, 1956.

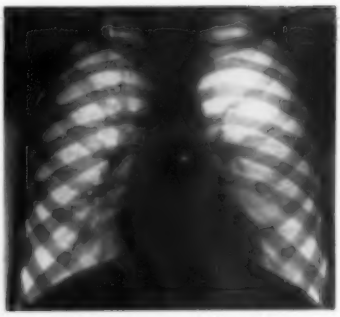

Fig. 5. Postero-anterior view. December 18, 1956.

febrile bronchopneumonia of the right base; radiography revealed an infiltration of the lower half of the right lung and a hypertransparent view of the upper half of the left hemithorax, with emptiness of the hilar horn, especially evident on tomography (Fig. 1). Bronchography (Fig. 2, 3) revealed that the left upper bronchial tree was thin, with a downward displacement of the upper lobar bronchus which became more visible and showed an approximation of the lower branches of the same side. After recovering from the right bronchopneumonia, the child had no more respiratory troubles. Therefore any surgical intervention, such as the intervention suggested by Crenshaw[57], was put off. The x-rays of the lung, taken after one month (Fig. 4) and some months later (Fig. 5) demonstrated an unchanged persistence of the hypertransparency of the upper part of the left hemithorax.

SUMMARY

1. The characteristics of the syndrome known as "vanishing lung," lobar emphysema," "abnormal transradiancy of the lung" are described. 2. The pathogenesis is discussed. 3. One case, observed in a 6-year-old child, is briefly reported.

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MARTY, S.D.; BECHTEL, B.; WOOD, C. E.; Incontinentia Pigmenti, Report of a Family (Arch., Derm. & Syph.) 78:607, Nov. '58.

The authors report an entity, incontinentia pigmenti, which is heredofamilial. A family tree of three generations is presented, showing all the female members to have the disease. While this is a rare condition, pediatricians should be on the alert for the manifestations which are present early in the neonatal period. Four stages are described. (1) Linear and grouped vesicles on an erythematous base which rupture and tend to impetiginization. (2) An intermediate stage of linear verrucous lesion may be present. (3) The third or pigment stage, shows a reticulated pattern of pigmented macules, whorls, lines and patches. (4) A final stage may reveal no signs of the disease or slightly atrophic depigmented areas. Ectodermal and mesodermal defects may be associated. Cases have been reported in males.

H. H. GORDON, M.D.

HYPERCALCEMIA RELATED TO NEUROBLASTOMA

RONALD N. MACLEAN, M.D.*

Buenos Aires

The presence of a malignant tumor can be suspected when one is faced with non-specific manifestations such as asthenia, pallor, fever, jaundice, cerebral or ocular disturbances, hepatmegaly, which can be partly due to early metastases. These signs are common to many malignant tumors, and at times the difficulty arises in discovering the nature and the location of the tumor, especially when the clinical findings, x-rays, and laboratory investigations throw little light on the picture.

This paper is limited to the subject of hypercalcemia as the only positive data in a child with non-specific clinical manifestations and who later proved to have a neuroblastoma.

CASE REPORT

P.J.J. a 3 year old girl whose chief complaints when admitted were enuresis and inability to move out of bed because of apparent pain.

Parents are healthy, and one healthy male sibling of 1 year of age.

Patient was an active and alert child, healthy until 2 months prior to the onset of the present illness when parents noticed an area of alopecia on the right side of head. A pediatrician was consulted who diagnosed ring worm and treated her with fungacides. This treatment was carried out for a month with no noticeable improvement. She was then treated with radium therapy. Depilation was performed with 300r per area. 6 weeks after the initial treatment she received a second treatment to the incompletely depilated areas, 300r to each of the three areas were applied. The result was satisfactory after 3 weeks, leaving only an incomplete epilation in the vertex and temporal regions of head.

Parents state that 5 months ago, previous to the radium therapy, child complained of a pain under her chin and on sternum when washed, and a month later she complained on occasions of legs hurting when out walking. As her general condition, appetite and activity remained good, no attention was paid to these symptoms. Child

* Attending physician. Hospital de Clinicas. University of Buenos Aires.

was very restless following the first radium treatment, and 2 weeks later complained of slight pain under arms and refused to be lifted from her arm-pits. At this time her hair fell out from the right side of head and very slightly from the left. On the following days she became very irritable and easily upset. She occasionally awoke at night crying and could give no definite reason except at times she complained of a pain in the abdomen.

Following the second radium treatment the child showed increasing signs of fatigue and disability, was easily upset and impatient.

Two weeks before admission she complained of a slight stiffness of knees, refused to walk and later was put to bed complaining of generalized pain mainly in thighs, and running a temperature of 38 to 39°C. (100.4 to 102.2F). Seen by the pediatrician she was found to have a pharyngitis, and was put on procaine penicillin injections. During these days it was noticed that her right eye-lid at times would droop, and she began to pass urine in her bed. At times she would remain quiet and happy but seemed to suffer great discomfort when she was moved. She was eating much less than usual; her water intake was about the same. Her urine was observed to be very cloudy with a 'sandy' sediment.

She remained on antibiotics for one week, and was then brought to the British Hospital for admission.

PHYSICAL FINDINGS

The patient is a well developed and well nourished 3-year-old girl, lying in bed and looking moderately acutely ill. She becomes irritable and appears to suffer pain when moved, otherwise she is alert and cooperative.

Head shows complete alopecia excepting area on top and temporal region. Face expressive with slight pallor. Eye grounds: normal. E.N.T.: Pharynx moderately injected. Abdomen soft, no resistance or apparent pain to palpation. No tumors palpated. Liver smooth, edge 2cms. below costal margin on anterior axillary line. Spleen not palpable. Rest of physical examination not contributory.

LABORATORY FINDINGS

R.B.C. 4.350.000 Hemoglobin: 88%, 12.8 gm. W.B.C.: 8.200 Neutro: 70% Lymphocite: 28% Mon: 2% Platelet count: 350.000 proteinemia: 6.50 gm. Urea: 0.35 Calcemia: 15.6 gms./100 ml. Phos: 5.2 mgs/100 ml. Alk. Phosphatase: 28 mmol King Armstrong. Potassium: 16:20 mgs/100 mil. Chlorides: 560 mgs/100 ml.

Sed rate: 1st hour 67, 2nd hour 88. Coagulation time: 6 minutes. Bleeding time: 2 minutes. Urinalysis: (catheterized) Alk. 51 gr.: 1005 Alb: neg. Glucose: neg. Acet: nge. Urobiline: neg. Ph: 8. Amorphus phosphates in abundance Urine culture: sterile. Bence-Jones protein in urine: negative. Lumbar tap: normal fluid. Sternal marrow puncture: negative. Vollmer tuberculin: negative.

X-RAY FINDINGS

X-rays of bones show some medullar shadows at the lower end of the femur and about the skull. These faint shadows are not exactly 'punched out', as they have not clearly limited edges within the marrow, nor is there rarefying of the bone trabecullae. Myelomata is ruled out with Bence-Jones proteins negative. These x-rays together with the high blood calcium suggest the possibility of hyperparathyroidim or a metastic lesion. (F. van Domselaar, M. D.)

A barium enema showed a distended atonic colon. An intravenous urogram showed a normal functioning right kidney, and an obstruction in the left uretro-pelvic angle with normal filling pelvis.

The cystoscopic examination showed a normal bladder. The ureteral orifices were normal; on both sides the urine flowed normally.

An ascending pyelogram was performed on the left side without meeting any obstruction. The pelvis was filled with dye showing a normal pelvis and calyx. The catheter was removed and another picture showed the pelvis emptying normally, ruling out obstruction. (Ernesto Donnelly, M.D.)

COURSE

On admission child was afebrile, and during the first few days showed no change in her general condition. Partial ptosis of her right eye-lid was noticed on one morning.

On the 4th day, following the cystoscopy, she ran a temperature of 39°, (102.2F) became very listless and appeared acutely ill. She was given terramycin 200 mgs. every 8 hours and 2 days later she was bright and happy and walked on her bed, not complaining of any pain.

On the following days her general condition got worse, and on the 12th hospital day she awoke with a bilateral convergent strabismus which persisted. That same evening a clot of blood was noticed in stools. Repeated laboratory tests showed an increasing anemia and the persisting hypercalcemia.

On the 14th hospital day, a tumor about the size of an olive was palpated deep in the left paravertebral region, in the epigastric zone. The child's general condition continued to decline, she showed marked loss of weight but continued alert and cooperative and only suffering pain when moved.

Three days later an exploratory laparotomy was performed in view of the clinical findings and the palpation of a tumor.

OPERATION

(Alberto Laurence, M.D.) Left paramedium incision. The gastrocolic oementum was divided widely exposing the anterior surface of the pancreas. On the anterior surface of the body of the pancreas a round purplish tumor the size of a pea is found. It is enucleated entirely for a frozen section, the biopsy showing definite malignancy. There is a large lobulated tumor extending along the left side of the vertebral column both above and below the route of the meso-colon, which is undoubtedly impossible to eradicate entirely. A large lobule the size of a cherry is removed by biopsy, the remaining portion of the tumor is entirely fixed in its paravertebral position medially to, and extending both above and below the limits of the kidney.

PATHOLOGY REPORT

(O. C. Croxato, M.D.) The specimens sent for examination consist of a pancreatic nodule and a node measuring 2 x 3 cms. The latter is soft and its out surfaces show a replacement by an amorphous substance with yellowish areas. Smears suggest neuroblastoma.

Microscopical examination: the sections of both the pancreatic nodule and the larger tumor show that they are formed by small cells which in areas have a fusiform appearance, chiefly rounded with a small amount of cytoplasm and a large hyperchromic nucleus. There are many necrotic areas and some atypical rosettes. The cells are separated by eosinophilic fibrillar material, and connective strands are also seen forming spaces which contain the tumor cells. Diagnosis: neuroblastoma.

POST OPERATIVE COURSE

Following the operation the child received a blood transfusion. Radiumtherapy was started on the 2nd postoperative day, and was continued till shortly before the child's death one month later.

DISCUSSION

Besides the non-specific manifestations in this child, the hypercalcemia was the one finding from which to orient the diagnosis. Perhaps more value could have been placed on the apparent obstruction in the left ureter which was possibly caused by the tumor, but which was ruled out because of the negative ascending pyelogram. The palpation of the abdomen in this child was easy as she was always cooperative. It may be advisable in these cases where there is a suspicion of metastatic lesions to anesthetize the patient for a more thorough examination with completely relaxed muscles.

With the presence of hypercalcemia, the causes which can give this finding must be kept in view for a differential diagnosis.

1) The mobilization of calcium from its storehouse in the bones with an increase of serum calcium is a constant finding in hyperparathyroidism. This is accompanied by a decrease in serum P, increased serum phosphatase activity and urinary P and Ca.

2) The serum calcium concentration is usually normal in most cases of primary and metastatic neoplasms of bones. However, high values have been observed in cases of extensive metastatic involvement of bones. In our case the X-ray findings of bone lesions were few. When there is hypercalcemia following neoplasms of bones, the values for serum P and phosphatase activity are usually within normal limits, except in osteogenic sarcoma in which the serum phosphatase activity may be increased.

3) Hypercalcemia has been reported in about 50% of cases of multiple myeloma. This does not seem due to a state of hyperparathyroidism as most frequently there is no skeletal abnormality, the P is not decreased and the serum phosphatase activity is normal. This tumor is rare in childhood, and the negative Bence-Jones protein test is an aid in discarding this diagnosis.

4) Other rare cases in which hypercalcemia can be encountered are advanced nephritis, administration of excessive amounts of vitamin D, and occasionally in leukemia and polycythemia vera.

SUMMARY

The case of a child with hypercalcemia due to neuroblastoma is presented. The differential diagnosis of hypercalcemia is discussed.

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ACUTE PEPTIC ULCER AFTER CEREBRAL TRAUMA IN CHILDREN

SAMUEL ROSNER, M.D., F.I.C.S.*

New York

Two children with severe trauma to the brain suffered from acute hemorrhagic peptic ulcer with onset of hemorrhage within a few days of the cerebral trauma.

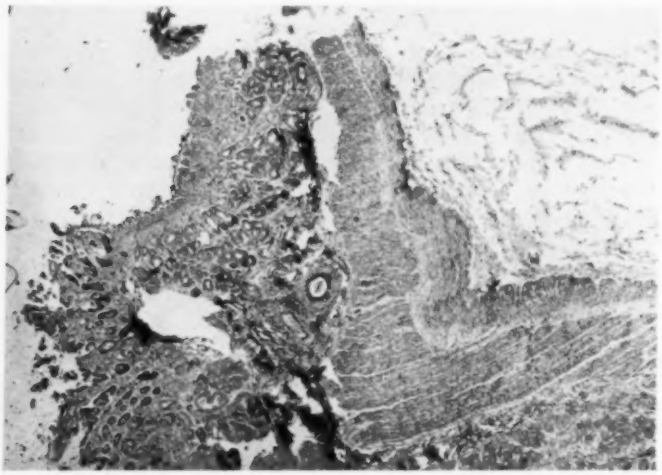
Cushing[1A, 1B] has drawn attention to the violent contractions of the stomach that result from operative interference with tumors which involve the basal part of the brain. Such contractions seem to cause hemorrhage and ulceration in the stomach, often with perforation and fatal result. This confirms the idea of Rokitansky who many years ago put forward the statement that gastric ulcers are caused by a nervous disturbance. Cushing[7] recognized the dependence of these effects upon the stimulation of para-sympathetic centres in the hypothalamic region. There, the three nuclear masses, the supra-optic, the tuberal and the posterior or paraventricular, can be distinguished, the tuber nuclei being distinctly para-sympathetic in their relations while the posterior or paraventricular are sympathetic. Cushing showed that injection of pituitrin or of pilocarpine into the ventricle produces the most violent sweating and peristalsis of the stomach with vomiting, evidently a para-sympathetic response. He thought the effect analogous to that produced by the secretion of the adrenal medulla upon the sympathetic. Watts and Fulton,[2] have since, by producing lesions in the tuber nuclei in monkeys, induced profound gastro-intestional disturbances with gastric and duodenal erosions and perforation, although similar lesions in no other part of the nervous system had this effect. The subject is discussed in Fulton's[3] paper on the hypothalamus and visceral mechanism.

There have been many reports of gastro-intestinal disturbances following cerebral injuries both in man and in animals[4,5]. Acute ulceration of the gastric mucosa may develop within a few hours of a cerebral injury, especialy one affecting both cerebral hemispheres. In the cat following removal of both motor areas, a greater persistency and constancy of strength of gastrict peristalsis is noted with a considerable increase in resting contraction.[6]. Kennard[6] has written, 'The accumulation of evidence on cortico-autonomic function

*Director of Neurology and Neurological Surgery, Parkchester General Hospital.

points to a focal representation on the cortical surface which is very similar to that of the somatic representation and which exists to some degree in the cat, dog, monkey, and man."

Cushing[8A, 8B] pointed out that certain brain tumors may end with a perforated gastric ulcer, and that the same is sometimes true of operations on the cerebellum and of intracranial injury in the newborn. He suggested the presence of a parasympathetic center in the hypothalamic region from which tracts relay back with cranial



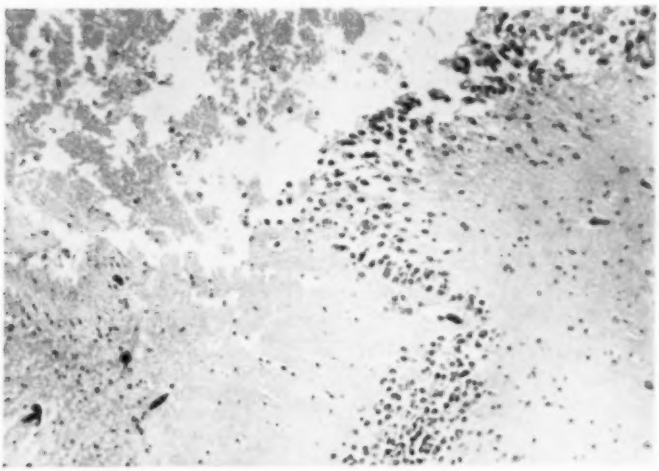
Acute duodenal ulceration following cerebral trauma.

autonomic stations in the mid-brain and medulla, of which the vagus nucleus is the most important. Experimental lesions of these tracts from the hypothalamus to the vagal center has been shown to cause gastric erosions, ulcers and even perforation, probably due to nervous impulses leading to temporary ischaemia and finally necrosis of the gastric mucosa. Two illustrative case reports follow:

Case No. 1: — K. V., male, colored, age 6½ years. Was struck by an automobile and rendered unconscious on Oct. 4, 1957. Bilateral subtemporal craniotomy was carried out. The child suffered bilateral subdural bleeding with brain laceration. The patient had suffered from grand mal epilepsy from shortly after birth. The brain laceration was in the right fronto-temporal area. About the 12th of October, the child began to pass bloody stools. Despite frequent transfusions he died on the 18th of October. Autopsy showed

that there had been a fracture of the pelvis, acute ulceration of the stomach and brain injury. Microscopic Examination showed hemorrhage into the basal ganglia bilaterally.

Case No. 2: — R. C., male, white, age 20 months. Was diagnosed as having cerebral palsy with mental defect. Bilateral craniotomy on Oct. 2, 1958 was carried out and a venous varicoscity



Area of cerebral trauma.

above the right Sylvian fissure and a large arterio-venous 'lake" [9] at the lower angle of the left Sylvian fissure were removed by electrocautery. The child regained consciousness post-operatively, played wtih his parents and ate well. On Oct. 6th, the child began to pass tarry stools, Shortly thereafter, he exhibited vomiting of blood and passed massive bloody stools. Despite adequate blood transfusions the child died on Oct. 6th. Pertinent autopsy findings were (1) Porencephaly involving the left fronto-parietal area. (2) Acute duodenal ulcer.

SUMMARY AND CONCLUSION

Two cases of acute hemorrhage from a peptic ulcer following trauma to the brain are described. In one patient the injury was confined to the cortex bilaterally. In the other the cortex showed

laceration on the right side but microscopic examination showed injury to the basal ganglia of a hemorrhagic nature.

These two cases confirm the experimental and clinical observations described in the foregoing portion of this article. However, the purpose of this article is to present the findings in children which in no way differ from the findings in adults.

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1882 *Grand Concourse, Bronx 57*

THE TREATMENT OF INTERTRIGINOUS ERUPTIONS (diaper rash) AND INFANTILE ECZEMA

HARRY R. LITCHFIELD, M.D., F.A.C.P.*

New York

Previous experiences with a variety of ointments, lotions, or powders in the treatment of intertriginous eruptions have in many instances been found to be far from satisfactory, until the introduction of steroids. The purpose of this paper is to analyze our findings with this newer type of therapy.

METHOD OF STUDY

The present study was undertaken in two maternity hospitals averaging about 3500 babies a year, and in out-patient clinics. The newborns were observed for intertrigo, usually occurring in the newborn almost only in the genito-gluteal region; the other localizations in the axillary folds, such as the neck, are only seen exceptionally and at a later period in infancy.

The ultimate cause is almost invariably irritation from the stools and urine. Frequent stools in this first period of life (from the second half of the first week) with redness and wetting are a common cause for dermatitis (diaper rash).

It is by no means always sufficient just to change and clean the newborns before feeding them. They require drying of the areas.

Apart from the unpleasantness to the infant, the wet surface robbed of its upper layers of epidermis forms a convenient portal for the exciting cause of this condition.

It must be emphasized that the data (as presented in the accompanying charts) reflects a study of only a short period of time in the lives of a relatively small number of infants. The results, however, appear to suggest a trend in the newer therapy for intertriginous eruptions and infantile eczema.

METHODS OF APPLICATION

Best results are frequently obtained by applying locally a thin layer of Pantho-F® as soon as any simple redness appeared. This

* Director of Pediatrics — Brooklyn Women's Hospital
— East New York Dispensary
Senior Attending — Beth-El Hospital

method employed in our nurseries was extremely effective and prevented further skin irritations.

In several of our cases the skin irritation was caused by the ammonia produced by the action of urinary urea by gram positive bacterium ammoniages. Here the addition of d-1-methionine (mg. 5) added to two or three formulas a day for a few days helps in clearing up this condition. It is truly a deficiency of methionine that results in an ammonical urine. d-1-methionine with local application of Pantho-F quickly clears up the irritation, if the condition has not progressed too long, as in one case where a local infection resulted and an antibiotic was indicated.

Diaper rash before treatment

Clearing of rash with Topical Pantho-F and D-L Methionine

PHARMACOLOGY OF THE CREAM

Hydrocortisone itself has no antibacterial activity. Its combination with mild antieczematous antibacterial agents is therefore in the treatment of the delicate skin of the newborn. Panthenol has been found to exert an antibacterial, anti-inflammatory and antieczematous effect when incorporated in a cream. (Kline and Caldwell, etc.[2].) Hydrocortisone has a non-specific antipruritic and anti-inflammatory effect when applied to the skin. The combination of these two agents was found to be a very effective one, producing effects which were more than the addition of the two agents would produce singly. This has been shown to be a synergistic effect and the hydrocortisone can be reduced to a very low percentage in this combination with the same therapeutic effectiveness as when the steriods are used in higher concentration, 1%, 2.5%. This low percentage of hydrocortisone obviates the possibility of causing systemic effects. Livingood et al[1] have shown that systemic effects could be produced from topical application.

CHART I.

OBSERVATIONS ON INTERTRIGINOUS ERUPTIONS

(DIAPER RASH) AND

INFANTILE ECZEMA TREATED WITH PANTHO-F* 0.2%

TYPICAL CASES

NAME	AGE	AREA OF INVOLVEMENT	TREATMENT AND RESULTS
CB	4½ mos.	Fine vesicular rash resembling miliaria rubra on face — 3 weeks duration.	5-23-57 Cream applied t.i.d. 5-30-57 Condition cleared Rash completely disappeared on 7th day.
S.R.	3 mos.	Intertrigo in both groins — 1 month duration.	3-21-57 Cream applied t.i.d. Condition cured in 2 weeks.
L.T.	2½ mos.	Inflamed and crusted urinary meatus in male infant.	5-23-57 Cream applied several times daily. 5-30-57 Condition cleared. Perfect healing in 5 days.
G.S.	6 mos.	Presented a hemorrhagic vesicular rash (size of pin head) covering the greater part of the scalp—1 week duration.	4-16-57 Cream applied t.i.d. for 1 week — failed to show improvement. The condition then spread to the upper and lower extremities. Consultation with Dermatologist. Opinion — Infantile Eczema—advised Soybean (vegetable milk). Dietary changes cleared the involvement in 10 days.
R.D.	3 mos.	Dermatitis of both cheeks — 7 days duration — probably due to rubbing on bed clothes.	3-28-57 Cream applied t.i.d. Rash disappeared after 4 days.
S.M.	11 wks.	Had a papular eczematous appearing rash on both cheeks and chin.	5-16-57 Cream applied t.i.d. Complete disappearance of rash after 4 days. No recurrence 6 weeks later.
D.W.	8 mos.	Ulcerated intertrigo of buttocks of 3 days duration.	4-2-57 Cream applied t.i.d. 4-9-57 Returned to clinic. The lesions had cleared in 3 days.
D.R.	5 mos.	Severe intertrigo in both groins —5 days duration.	3-28-57 Cream applied t.i.d. Cured in 2 days.
J.B.	7 mos.	Presented an eczematous lesion of right external ear.	4-11-57 Cream applied t.i.d. 6-10-57 Eczematous lesion was completely healed. No dietary change required.
F.K.	8 mos.	Contact dermatitis (Dermatitis Venenata) caused by plastic bib which irritated the posterior neck. Duration was 2 weeks before treatment with Pantho-F was instituted.	6-13-57 Cream applied t.i.d. 6-20-57 Almost complete disappearance of dermatitis after 1 week treatment.
J.S.	3 mos.	Had 7 pustular lesions over occipital scalp. Duration 7 days.	6-20-57 Cream applied t.i.d. 6-27-57 All pustules cleared. New ones appeared which were similarly treated with gratifying results.
S.G.	5 wks.	A severe case of milium covering cheeks and forehead of 2 days duration.	3-28-57 Cream applied t.i.d. Condition was cleared after 2 days treatment.
H.Z.	12 mos.	Eczema of both hands — of 5 months duration.	7-18-57 Cream applied t.i.d. 7-31-57 Showed a marked improvement. Advised to continue Pantho-F. Did not return for further observation.
C.A.	4½ yrs.	Eczema present circumorally and on cheeks since infancy.	4-17-57 Cream applied t.i.d. 4-24-57 Child's skin appeared perfectly normal. This was a chronic eczema of long standing.

* Pantho-F℞ — supplied through courtesy of U. S. Vitamin Corp.

NAME	AGE	AREA OF INVOLVEMENT	TREATMENT AND RESULTS
L.W.	10 yrs.	Has had recurrent involvement of eczematous lesions of face, arms, and hands. This time half the palm and the small finger were involved—duration from May 20 to June 22, 1957.	6-22-57 Cream applied t.i.d. 6-26-57 Within 4 days hand appeared almost normal. This was the first time the patient got rid of her eczema so quickly. It used to take months before the eczema would disappear. 10-3-57 Revisit to clinic showed no involvement and condition was excellent during the entire summer.
H.M.	8 wks.	Marked erythema of the labia majora, with excoriation.	4-15-57 Cream applied t.i.d. 4-22-57 No improvement. Probably a local skin irritation with a urine of strong ammoniacal odor. Non-responsive to Pantho-F but cleared with aureomycin ointment and d-l methionine 5 mg. (one capsule 3 times daily in formula. Condition then cleared. This was a case of methionine deficiency.
K.S.	2 mos.	Seborrhoea of scalp and ear lobe. Duration 2 weeks.	7-10-57 Cream applied t.i.d. Cleared after 4 days treatment with Pantho-F.
R.L.	5 yrs.	Eczema present over elbow and the posterior aspect of both knees. Duration 1 year — started May 1956.	5-24-57 Cream applied t.i.d. 6-17-57 Lesion had disappeared. 7-25-57 Condition cleared.
I.C.	1 mo.	Fine papular dermatitis of both cheeks.	5-22-57 Cream applied t.i.d. 5-27-57 Skin condition cleared.
M.H.	6 yrs.	Prurigo—involving extensor surfaces of arms, wrists, elbows and legs — accompanied with itch. The condition started in early infancy (at 4 months of age).	5-11-57 Cream applied t.i.d. 5-18-57 No results—advised to continue same treatment. 5-27-57 Conditioned unchanged. Pantho-F stops the itch and improves the condition. However, there is a return of symptoms and signs on cessation of treatment. Referred to Dermatologist. Opinion — Allergic food manifestation.
T.R.	10 mos.	Eczematous papular dermatitis—duration 5 weeks.	5-27-57 Cream applied t.i.d. 6- 3-57 Marked improvement and disappearance of lesion. Condition cleared in 7 days.
M.A.	18 mos.	History of redness and moderate swelling of the labia majora and dermal involvement of groins. Occurred shortly after birth. Various ointments failed to give results. The left elbow presented an area of an eczematous appearing lesion which we feel was dermatophytid in nature.	4-27-57 Cream applied t.i.d. 5- 6-57 All lesions cleared in 10 days, as observed on this date. Pantho-F cleared the elbow involvement in 5 days. The mother remarked, "For the first time in 18 months the child's groins and privates look well and healthy."
M.A.	3½ wks.	Diaper dermatitis over lower abdomen and both thighs of 10 days duration.	3-30-57 Cream applied t.i.d. 4- 4-57 Condition cleared.
B.B.	12 yrs.	Ears were pierced for earrings. Infection set in at points of puncture. Sulfa and other antibiotic ointments failed to clear the infection of 1 mo. duration.	6-18-57 Cream applied t.i.d. 6-22-57 Condition cleared. Clinically we felt this was a fungus and bacterial mixed infection.
J.K.	12 mos.	Anal pruritus with intertrigo treated 2 months with various ointments without improvement.	5-2-57 Cream applied t.i.d. 5-9-57 Condition cleared. There was complete disappearance of all signs and symptoms in 7 days.

PANTHO-F 0.2% CREAM

	Total Cases	Healed	Remarks
Intertriginous Eruptions	30	24	86%
Infantile Eczema	16	13	81%
Other (chronic) Dermatitis	1	0	referred to dermatologist

CONTROLS

	Total Cases	Healed	Remarks
Cod Liver Oil Ointments			
Intertriginous Eruptions	12	4	33%
Intertriginous Eruptions	5	4	lasted several weeks 80%
Powders (zinc, etc.)			
Infantile Eczema	10	1	10%

SUMMARY

Present day ointments per se are not very suitable for the treatment of intertrigo, or for that matter, infantile eczema. True eczema, in its various forms, does not strictly speaking occur in the early newborn period. However, allergic manifestations occuring on the face (cheeks) due to early introduction of wheat cereals, milk albuminates, or fresh orange juice, may present themselves for therapy. Here, the response to Pantho-F in the treatment of atopic dermatitis, allergic skin conditions and a variety of inflammatory conditions, including infantile ezcema, was prompt clearing of the skin in a large percentage of cases as compared with controls.

CONCLUSION

Pantho-F Cream 0.2%, a combination of panthoderm cream incorporated with 0.2% hydrocortisone, has proved to be a reliable, effective and safe preparation for the treatment of infantile eczemas of all types and especially rapidly effective in the treatment of intertriginous eruptions and "diaper rash" of early infancy.

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Current Literature

Edited by

MICHAEL A. BRESCIA, M.D.

ULTRAMICRO METHODS FOR CLINICAL LABORATORIES, by Edwin M. Knights Jr., M.D., Roderick P. MacDonald, Ph.D., Jann Ploompuu. Cloth bound, 128 pp., 18 Fig., $4.75, New York, Grune and Stratton. 1957.

This book is primarily intended for the clinical pathology laboratory. It offers a clear, concise methodology for the determination of ultramicro methods of chemical analyses. Chapters one, two and three are of special merit, offering to the novice and experienced invaluable information on the setting up of an ultramicro laboratory, the special equipment needed and the methods used in securing and pipetting of blood samples.

The specific tests are arranged in an orderly, easy to use fashion making the book a must for the laboratory work bench.

The pitfalls as well as the advantages of ultramicro technics are admirably presented.

This book is recommended, without qualification, for the clinical pathology laboratory. JAMES E. MCNALLY, B.S.

TUBERCULOSIS IN WHITE AND NEGRO CHILDREN. Vol. I. The Roentgenological Aspects of the Harriet Lane Study. By Janet B. Hardy, M.D. Cloth. Illustrated. Pp. 122. Price $7.50.

Vol. II. The Epidemiologic Aspects of the Harriet Lane Study. By Miriam E. Brailey, M.D., Dr. P. H. Cloth. Pp. 103. Price $4.50. Harvard University Press for the Commonwealth Fund. Cambridge, Mass. 1958.

This carefully documented report tells of the outcome of tuberculous infection among a group of underprivileged white and negro children between 1928 and 1950 in the days before specific therapy was available for this disease.

The first volume contains reproductions of a series of x-rays typical of the clinical situations handled. There is also a section describing technics of bronchoscopy and bronchography. The importance of a thorough knowledge of the normal anatomy of the bronchial tree and the segmental pattern of the lung is stressed because of the importance of endobronchial disease in producing many of the parenchymal manifestations typical of primary infection.

The second volume presents the actual findings of the study in great detail and includes an extensive and interesting bibliography. This review can only skim over all the material making up this part of the report.

The study is based upon the follow-up of 437 white and 892 negro children, patients of a pediatric tuberculosis out-patient clinic.

Supervision by the clinic began with the discovery of tuberculous infection. 44% were under 3 years of age when this was diagnosed, the others were anywhere from 3 to 14 years old. Within each racial group the distribution of the ages of the children on admission was comparable and the sexes were equally represented.

The younger group of children were found, on initial examination, to show more frequent and more extensive x-ray evidence of tuberculous disease than the older ones. To what extent this represents age-specific resistance to primary infection could not be determined since all children were already infected when admitted to observation and in no case was there definitive knowledge as to the age at which this had occurred. However the lesions seen among the younger children were for the most part typical of freshly acquired infections whereas many of the older ones had findings suggesting infection of considerably longer duration. The author feels that at least some of the observed age differences can be attributed to the fact that a number of the older children had passed through the initial acute phases of their infection some time before coming under observation.

In both age groups, on initial examinations, x-ray evidence of disease was more frequent and more extensive among the negro children. This racial difference was greater in the children who were 3 years old or more when supervision began.

White children remained under observation for an average of almost 14 years and the negroes for over 11 years. Sufficient numbers of children were followed for long enough periods of time to enable calculation of mortality rates by the life-table method in terms of a 15 year experience. These rates were closely correlated with the initial x-ray findings within each race and age group. The sex of the child had no influence on the probability of death from tuberculosis, nor was it possible to demonstrate any relationship between fatal outcome and continuing household exposure to tuberculosis.

The situation was somewhat different when, again by life-table methods, the rate of appearance of reinfection tuberculosis among these children was calculated. Here, among the negroes, there was a strong suggestion that continuing family exposure increased the

likelihood of developing this complication. As far as the white children were concerned, reinfection tuberculosis occurred in only two instances in the whole series yielding a rate 1/10 of that which was found among the negroes. The author feels that part of this favorable picture is due to the fact that very few children were followed through late adolescence and into early adult life. It is at this period that previously infected white population tends to develop reinfection disease whereas in negroes this complication begins to appear in the early teens. Negro girls were 5 times more likely to break down than were the boys although the case fatality rate was about the same, about 50%.

The question of the origin of chronic progressive pulmonary disease is discussed at some length. It is becoming increasingly obvious that previously infected children are not the only source of this type disease. Experience in accumulated Scandanavian countries shows clearly that when primary infections are acquired in adolescence or early adult life the disease produced is much more apt to be phthisis than when the first infection occurs in infancy or early childhood. The trend of tuberculin surveys over the past decades indicates that the age when first infection occurs is continually rising in both white and negro populations. We cannot estimate the relative importance of recently infected abolescents and young adults compared to that of individuals infected in early childhood as contributors to the current prevalence of chronic pulmonary tuberculosis. But the implication is plain. A modern program for the control of tuberculosis must include a search for recently infected individuals in this age group through widespread and repeated use of the tuberculin test in the doctor's office and in the community at large.

ANN P. KENT, M.D.

PATHOPHYSIOLOGY IN SURGERY: By JAMES D. HARDY, M.S., (Chem.), M.D., F.A.C.S., pages 704, cloth, The Williams and Wilkins Co., Baltimore, Maryland, 1958, Price $19.00.

The material in this book is drawn from the author's experience and from other reference sources. Written in text-book form, pathophysiology in surgery is clear, comprehensive and concise. The subjects discussed comprise the standard work-a-day physiology of surgical practice. It entails anatomy, chemistry, internal medicine, microbiology, pathology, physics, physiology and surgery in a functional way. The book represents constructive material, a fusion of "medical" with "surgical" physiology. The surgeon becomes much more of an internist in his day to day surgical practice. The book

represents and emphasizes the fundamental unity of the human organism which is achieved through three major co-ordinators: 1. The central nervous system. 2. The circulatory system. 3. The endocrine system.

The contents of this book are divided into 21 chapters. The first ten chapters deal with the physiology of injury; fluid physiology; surgical nutrition; connective tissue; wound healing and homotransplantations; thermal burns; the etiology spread, and control of cancer; radiation and radioactive isotopes; surgical enzymology; and the biology of aging. These chapters serve as an introduction to the consideration of diseases of the various organs which follows in the next eleven chapters.

The eleven chapters dealing with surgical diseases of different organs begins with a brief survey of normal physiology and then proceeds to pathophysiology and the therapeutic problems involved. Numerous illustrations, photographs and charts facilitate reading and comprehension of the subjects with references following each chapter for those interested in further details.

I recommend this book to all general surgeons, general practitioners and medical students interested in surgery. It will certainly add to his armamentarium of surgical therapy in private practice.

Joseph M. Covelli, M.D.

Cleft Palate and Speech: 4th Ed. By Muriel E. Morley. Cloth. Pp. 271. Illustrated. Price $6.50. E. & S. Livingstone, Ltd., Edinburgh and London 1958. In the U.S.A. The Williams and Wilkins Co., Baltimore, Md.

This small but excellent volume will be of great help to the student of speech therapy or to the therapist. The author briefly outlines the embryological aspects of clefts of lip and palate as well as the anatomy of the normal palate. There is a chapter devoted to the surgical corrections of the clefts. Although the speech therapist should be acquainted with the various procedures used in correcting the clefts surgically, the details of the surgical procedures could very well be curtailed or eliminated.

The chapters devoted to the type of speech characteristic of these patients and the methods of correcting the speech are well done. This book should be of interest to those who concern themselves with the speech of the child with cleft palate. M.A.B.

UBER DIE ERBLICHKEIT DES NORMALEN ELEKTROENCEPHALOGRAMMS. VERGLEICHENDE UNTERSUCHUNGEN AN EIN- UND ZWEIEIIGEN ZWILLINGEN. By Dr. F. Vogel. Paper. Illustrated. Price $2.30. Georg Thieme Verlag, Stuttgart 1958. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y.

This monograph begins with the review of previous studies concerning the EEG in monozygotic and heterozygotic twins. Following this, the author reports on very careful electroencephalographic studies on 208 pairs of twins of which 110 were monozygotic and 98 heterozygotic. The ages vary from below 9 years to about 19 years, with the majority of the cases between 9 and 19. Records from the two twins were taken simultaneously with 12 electrodes, usually with a 16 channel machine. Electroencephalograms were taken with the patient awake, during sleep, with the eyes open and closed, during hyperventilation and induced oxygen deficiency.

The electroencephalograms were evaluated very carefully. It was found among other results, that the basic rhythm is the same in all pairs of identical twins, while it differs in ⅔ of the heterozygotic twins. In monozygotic twins, the alpha index shows no greater difference between the twins than usually found between hemispheres of one and the same person. In heterozygotic twins the difference is much greater. No great difference was found between slow activity in monozygotic and heterozygotic twins. The average amplitude is very similar in monozygotic twins, different in heterozygotic twins. While the EEGs of all monozygotic twins can generally be considered as being practically identical, this is the case only in 17 of the 98 pairs of heterozygotic twins. The same similarity as found for the electroencephalograms of identical twins with the eyes closed at rest is found under a great variety of other conditions as e.g. sleep, hyperventilation.

The studies do not confirm previous statements which postulate a relationship between alpha index and amplitude to handedness.

The authors conclude that the characteristics of the EEG at rest and during sleep are in all details determined by heredity. Exceptions are extremely rare. The mateuration of the EEG in identical twins is simultaneous in both.

Everybody who is interested in the heredity of the EEG and who intends to do twin studies using the EEG, should be acquainted with this very careful and valuable study.

HANS STRAUSS, M.D.

FEBRUARY 1959

KULCZYCKI, L. L. AND SHWACHMAN, H.: Studies in Cystic Fibrosis of the Pancreas. Occurrence of Rectal Prolapse. (New England Journal of Medicine 259:409. August 28, 1958).

Rectal prolapse is a common complication in cystic fibrosis, occurring in 22.6% of 386 patients.

This complication is most frequent between 6 months and 3 years of age and is rarely encountered after 5 years of age. There is no sex predominance.

In 16 patients, rectal prolapse was the presenting complaint of cystic fibrosis. In 3, the diagnosis of cystic fibrosis was made 7, 8 and 10 years after this initial complaint.

Rectal prolapse may occur as an isolated event, as in 8 patients, or may be a recurrent complaint extending over a 5-year perior, as noted in 5 patients. In 48 patients this symptom lasted longer than 6 months.

The institution of dietary therapy, primarily a high-protein and a reduced fat intake combined with pancreatic replacement therapy, combined with antibiotics to control pulmonary infection, will usually correct many of the factors responsible for rectal prolapse. The earlier the diagnosis of cystic fibrosis, the less likelihood of rectal prolapse. Surgical correction is rarely, if ever, indicated from our experience to date.

In any child with rectal prolapse of unknown etiology the diagnosis of cystic fibrosis should be considered, and in addition to a carefully taken history, appropriate laboratory tests such as the stool trypsin test, the chloride imprint test, determination of sweat electrolyte concentration or the more difficult duodenal-fluid assay for pancreatic enzymes should be performed to confirm or rule out this disease. Authors' Summary

FOERSTER, D. W. AND SCOTT, L. V.: Isolation of Herpes-Simplex Virus From a Patient with Erythema Multiforme Exudativum (Stevens-Johnson Syndrome). (New England Journal of Medicine 259:473 Sept. 4, 1958).

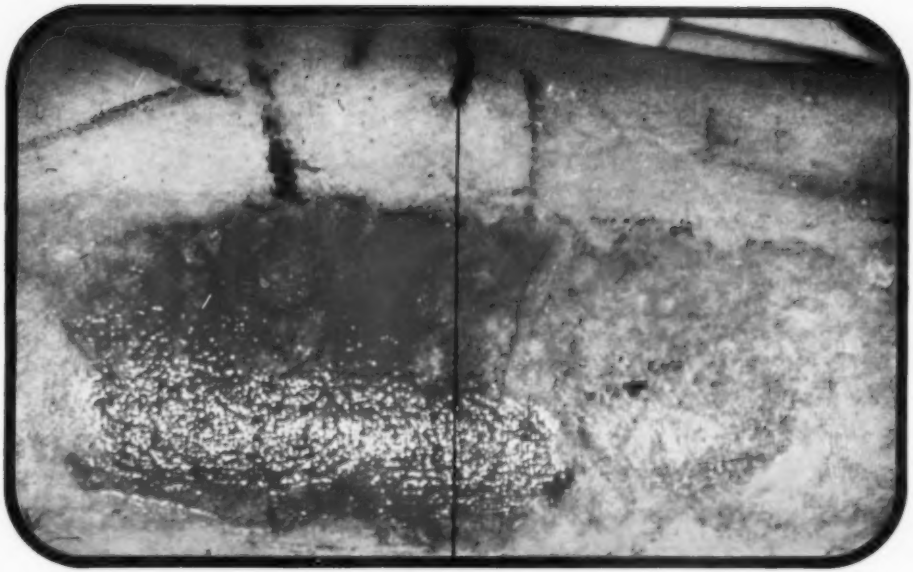
A case of erythema multiforme exudativum is reported in which herpes-simplex virus was isolated from vesicular fluid and identified by serologic technics. A significant rise in antibody titer to the virus was demonstrated. Previous studies in which this virus was implicated in the pathogenesis of erythema multiforme exudativum are review, and its potential role as the etiological agent is discussed.
Authors' Summary

ADVANCES IN PEDIATRICS: Volume X. Edited by S. Z. LEVINE, M.D. Cloth. Pp. 362. Price $9.00. The Year Book Publishers, Inc., Chicago, Ill. 1958.

The last of the "Advances" has maintained the high standard which was characteristic of the previous volumes. The tenth volume contains some very timely and significant presentations. The articles concerned with the treatment of tuberculosis in childhood and staphylococcal infections in nurseries are especially pertinent.

The subjects contained in this volume are as follows: 1. Psychologic Principles in Pediatric Practice: the Pediatrician and the Sick Child, by Barbara M. Korsch; 2. Treatment of Tuberculosis in Childhood, by Edith M. Lincoln; 3. Convulsive Disorders in Infants and Children, by Samuel Livingston; 4. Prevention of Poliomyelitis by Vaccination, by Albert B. Sabin; 5. Staphylococcal Infections in Nurseries, by T. E. Shaffer, J. N. Baldwin and W. E. Wheeler; 6. Muscular Disorders of Childhood, by Frank H. Tyler; and 7. The Transfer of Antibiotics from Mother to Offspring, by Bo Vahlquist.

M.A.B.



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Jeffords, J. V., and Hagerty, R. F.: Ann. Surg. *145*:169, 1957.

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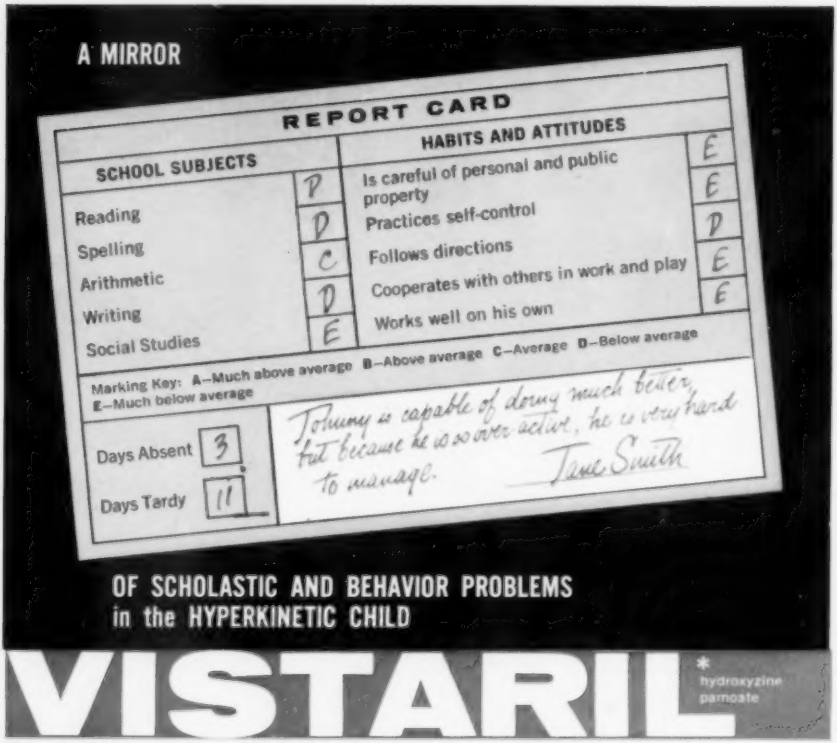

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REFERENCES: 1. Eisenberg, B. C.: Clinical Medicine 5:897-904 (July) 1958. 2. Ayd, F. J., Jr.: Med. Arts and Sciences 2:37, 1957. 3. Ayd, F. J., Jr.: New York State J. 57:10 (May) 1957.

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